The Madras Clinical Journal

Vol. XXIX January 1963 No. 7

CONTENTS

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The Madras Clinical Journal

JOURNAL OF THE MADRAS STATE BRANCH OF THE INDIAN MEDICAL ASSOCIATION
(WITH WHICH IS INCORPORATED THE "MISCELLANY")

Vol. XXIX January 1963 No. 7

THE CONGENITAL ANOMALIES AND THEIR CAUSATIVE FACTORS

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There are no rigid boundaries to differentiate anomaly from the normal. The body and its organs always display some individuality in size, form or architecture. Any competent anatomist is free to set his own standards of normality. However, when an organ oversteps clearly the more liberal interpretations of an acceptable range of variation, the condition is known as anomaly or malformation. Countinuous gradations connect the normal, slightly abnormal and severely abnormal. Most of the abnormalities occur in the early weeks of pregnancy. The acquired defects are to be contrasted against congenital anomalies. They appear secondarily as the aftermath of mechanical weakness or disease.

Incidence of anomalies:

The percentage of incidence of anomalies is found to be 1 in 12 of carefully done autopsies and 1 in 50 of live births. In aborted foetuses, the incidence of anomaly is higher. Internal anomalies are higher in percentage than the external anomalies.

Sex incidence: There are certain anomalies that are more common in males, eg., cleft-palate, pyloric stenosis, imperforate anus, and anomalies associated with the alimentary system. In females, anomalies of the hip, brain and skeleton are of a higher incidence.

Before going into the causative factors, that will produce anomalies, we will quickly run through the normal steps in the development of the human embryo.

We know fully well that the embryo comes out of a single unicellular structure, the ovum, after it is fertilised by the spermatazoa.

Many theories have been mentioned on the role of the spermatozoa in the fertilisation:

(i) The spermatozoa is supposed to remove a block that prevents the egg from developing into an embryo[1]. Probably it gives an enzyme, that unmasks certain important reactive groups in the egg and increases permeablility and cell respiration.

(ii) Any physical factor that will set into motion these activities is enough for fertilisation. Spermatozoa can be dispensed with, as we all know, in parthenogenesis of the lower animals; the female species give rise to offsprings without spermatozoa coming in the picture at all. Experimentally adult frogs have been produced out of the ova that have been pricked with a needle[2]!

So we see that the egg is all the more essential element in reproduction, and spermatozoa is not as indispensable and important. We do not know whether the status of the male will be further reduced by future experiments!

The embryo uses the following techniques for its developement:

1. The cell proliferation: It is said that 45 generations of cell proliferation are sufficient to produce the total number of cells of the adult theoretically.

2. Growth: The growth is by protoplasmic synthesis, water uptake, and intracellular deposition. In the development of an embryo all organs do not develop in the same speed. There is differential rates of growth in the human body, for example the human embryo has got a large head when compared to the body. This ratio of the head to the body decreases as the child grows.

The factors controlling growth:

1. Constitutional factor: This is an inherited quality in which there is a basic rate of cell division and growth for every animal kind.

2. The temperature: There is an optimum temperature which is most advantagious for growth.

3. Nutritional: Amino acids are very essential and there is an optimum intake of food. Even if one takes excess of food, growth may not be accelerated.

4. Growth promoting factors: These are non-nutritional.

(a) Embryonic factor: These are cytoplasmic co-enzymes present in the embryo that accelerate mitosis. Embryonic juices added to the tissue cultures make them grow well.

(b) Hormones: They are essential for growth.

(c) Vitamins: They act as chemical catalysts. B_{12} is essential for growth.

As the ovum grows in size and multiplies the number of its cells, it begins to arrange its cells in a definite order. It cleaves in a definite order, in a certain axis. That axis is maintained. This axis in future gives to it bilateral symmetry. The cells begin to segregate at convenient levels. Here some physical factors such as surface tension may help in sending the cells to their respective places, even though the genetic inherent characters direct them to go and take up the positions. They first arrange into three germ layers, the ecto-, meso-, and the endo-derms. After it has formed the three layers, the embryo prepares to form its organs.

In the development of an organ, that is organogenesis:

1. Cells have to migrate from one place to another.
2. They have to aggregate.
3. They have to grow in the local area.

4. They have to fuse with some other part in some areas.
6. Split in some areas.
6. They may fold or bend.

After undergoing all these processes, the shape of the organ comes.

There are yet some more processes before the development is complete. The cell or the organ has to differentiate. All cells of all germinal layers are alike first. But all cells of adult organs are not alike. So the primitive similar cells in the primary layer has undergone histological differentiation in the adult. This is called *histogenesis*. Every cell also undergoes a chemodifferentiation. Lastly they assume a functional differentiation (begin to function).

Integration: It is not enough if only the organs develop. They should begin to differentiate histologically, chemically and functionally. There must be coordination among themselves. This is integration. Integration is effected by: 1. Endocrine system. 2. Body fluids which act as carriers of endocrines. 3. Nervous tissue which conducts impulses.

We will like to know why an individual stops his growth at a particular time:

The following are the factors that are concerned in the arrest of growth.

1. There is antagonism between cell proliferation and cell differentiation. The cells which are highly specialised are incapable of division (proliferation), for example nervous tissues, brain, etc.

2. Ageing.

3. Cell destruction: There is always cell destruction going on, when there is cell proliferation (best seen in the case of growth and modelling of bones).

4. Hormonal interference: There is always an interaction of hormones (one hormone facilitates and the other hormone interferes).

Now we will like to see the factors that will guide or instruct or order the ovum to adopt all these techniques and grow into an adult:

Every living organism comes from a pre-existing living organism. Every cell comes from a pre-existing cell. All the vertebrates are built around a common anatomical plan. Darwin explains that each part of the body contributes sex cells and as the sex cells go to the next generation, they give representation. So son resembles father. Weillsman says (somatic cells) body cells do not contribute at all. Sex cells only contribute. He says there is a germplasm which is self perpetuating. It never dies, and it is a continuous stream, and the temporary body is built around it at every new generation. The body resembles its parents because the body developes around the same immortal stuff, that is the germplasm in the sex cells.

There are self perpetuating elements, the GENES. Genes are present both in the somatic and sex cells. The genes are the enzymic catalysing agents. They are present in the chromosomes of the cells. They are responsible for the hereditary characters. There is little evidence that cytoplasm of a cell may also play a role in heredity.

There are other factors also that help and modify development. They are the environmental factors:

1. Light.
2. Temperature.
3. Moisture.
4. Food.
5. Chemicals.
6. Drugs.

Heredity or the genetic influence is more powerful than the environmental influences. They are responsible for the structural, physiological and mental qualities.

Environment can do the following:

1. They can modify the developmental expression of inherited characters.

2. They can condition the genetic appearance.

3. They can alter the genetic constitution of the chromosomes.

4. They can create new heritable characters.

We find in the study of embryology of human beings, the embryo resembles in successive stages animals in successive order, for example the embryo of the human being at one time resembles the fish; in that respect it possesses gill arches. The presence of the gill arch is only transitory. The human embryo utilises much of the gill arch component for the development of the face and neck. This is an example of the theory of recapitulation in which an individual during the development passes through successive stages of series of ancestors from whom he descended. There are certain structures which are present in the lower animals which the human embryo has abandoned, for e. g. the tail. There are certain structures of the lower animals which the human embryo has organised during its development. This organ works as a stimulant for the further development of a permanant organ. If the former is blocked, the development of the latter is also blocked. For example in the development of the kidney, the human embryo organises first a kidney resembling that of a fish. This pronephros or the first kidney is the stimulant for the development of the next kidney—mesonephros which again is the further stimulant for the development of a permanant kidney.

Similar to the above example you can find in the development of an embryo, that presence of one organ induces the development of another organ. If the former fails to appear, the latter also will never develop. It is said that the first one, the "inductor" secretes a substance the "evocator" which comes into contact with the neighbouring cells, and stimulate them to form the organ[3]. These go in an order. The first one that appears is the primary organiser. (This in the case of frog is the chorda mesodern in the dorsal lip). If this is taken out and implanted in another belly of a developing gastula, a secondary embryo will develop in the wall of the implanted embryo. The chorda mesoderm has induced the undifferentiated cells in the wall of another embryo to develop into a full embryo.

The primary organiser induces the neighbouring cells. The neighbouring cells become an organ. This organ in turn induces its neighbours. They in turn become another organ, and in turn they induce their neighbours. This goes on as a chain, in an orderly manner and at correct timings.

The single cell ovum has multiplied, has employed so many techniques, has developed so many cells, it has ordered them to go to certain positions, has commanded them to form organs. It effects coordination between the organs and begins to function as a human being.

Anywhere from the stage of the single cell to the birth of a baby, mishaps may occur, leading to anomaly.

The egg; the single cell is totipotent. Its cell can give rise to any organ. Experiments have been done in which the ovum has been halved and allowed to grow. It grows into a full adult being.

When the egg has divided into blastomeres (but not arranged into the primary layers), if one of the blastomeres is damaged, the resulting embryo may not be affected. Other blastomeres grow up and take up the role that would have been played by the destroyed blastomere. The resulting embryo is not affected. It is normal without any anomaly.

So we find in the very early stage of the development even if a major damage occurs, serious result does not ensue. The embryo has got the potentialities to regulate its development. But as days go on and after the germ layers are formed, any injury to a certain group of cells will affect the future organs developed from them. The embryo at this time reaches the mosaic stage where work has been allotted to its cells. They do their work alone and can't take up the work of the other cells. They have lost their regulative capacities and have become determined.

Next we will proceed to see the different types of anomalies met with and enquire into the cause. Which has caused it.

1. Congenital agenesis: Here the primordium of a certain organ fails to appear at all and so leads to agenesis. Some organ is completely absent e. g. absence of kidney, arm, or radius.

2. Arrest of development: The primordium appears, but does not fully grow; it stops short of full growth. e. g. dwarfism, infantile uterus, etc.

3. Developmental excesses: The primordium appears and grows excessively e. g. gigantism. This may be general or local, e. g. extradigits, double-penis, and excessive hairiness.

4. Fusion: The primordium appears, grows and fuses with another primordium, e. g. horse-shoe kidney.

5. Failure to subdivide: Primordium appears and grows but does not subdivide into minor components of certain organs, e. g. syndactyly, fused fingers or toes.

6. Failure to consolidate: Different primordia appear and make up a single organ, usually in the case of certain organs. These primordia fail to fuse with each other to make up a single organ and this leads to accessory or multiple organs of the same type, e. g. accessory spleen or accessory pancreas.

7. Failure to atrophy: In the case of certain organs in the developing embryo, the primordia appears, grows and after a certain time it has to atrophy to form natural passages. If the concerned structures do not atrophy, anomalies result, e. g. anal membrane. (Persistence leads to imperforate anus.)

8. Incorrect migration: Certain organs during their development migrate from one place to another, e. g. kidneys, testis, ovary, etc. It may so happen that the migration may be defective or in excess, leading to anomalies like the undescended testis. (Abscence of testis in the scrotum in case of man, or the presence of ovary in the external genitalia in the case of woman).

9. Misplacement: Organs may be in unexpected positions, e. g. teeth in the palatine bones or transposition of the viscera.

10. Atavism: Here the development proceeds as usual, but it recapitulates its ancestors, e. g. azygos lobe of the lung. This is present in quadrupeds and may occasionally be present in man.

11. Atypical differentiation: The end product is not like that of a normal foetus, e. g. congenital tumours.

Congenital anomalies depend upon the viability of the ovum and the spermatazoa from which the embryo is derived.

The viability of the egg or the human ovum is only one day[4]. After that degeneration occurs. There is functional deterioration in eight hours, and structural deterioration in 24 hours. The life of the spermatazoa in the female tract is found to be 14 days. But the actual fertilizing time is suspected to be only a day or two[8]. Many of the congenital abnormalities are thought to be arising from the fertilization of an aged ovum or a fertilization by an aged spermatazoa.

The protoplasm of the eggs have survival abilities. The egg with very poor survival ability dies in the uterus. The human beings developed from an egg of average survival ability die in middle age, whereas human beings from an egg of great survival abilities live up to a ripe old age, provided the environmental conditions are safe. The longivity of a man depends upon the quality of the egg and the survival ability and the protoplasm of the egg he is derived from.

There is a critical period for every organ in the course of its development at which there is accelerated growth and differentiation. The organ is very much susceptible to injury at that critical period. Any injury at this stage will result in an anomaly of that organ. Every organ has got its own critical period. The time is different for the different organs.

The following can cause congenital anomalies:

1. Radiation effect: Well illustrated by Hiroshima and Nagasaki patients. This causes the mutation of the genes and thereby leads to congenital anomalies that can go on for generations.

Radiation may cause:

1. Microcephaly. 2. Mental-deficiency. 3. Micro-opthalmia. 4. Malformation of the extremities[5].

2. Chemicals: Thalidomide babies prove clearly how chemicals can cause abnormalities.

3. Mechanical factors: Factors like too little amnion may give rise to distorted babies.

4. Deficiencies: Vit. A deficiency leads to eye defects[6]. The soft parts of the body may not be well developed. Vit. B deficiency may lead to retardation and shortening of the skeletal formation. Vit. D deficiency may lead to bowing of bones.

5. Hormonal deficiency:

   Pituitary — Dwarfs.
   Thyroid — Cretins.
   Gonads — Infantilism.

6. Diseases:
   1. Syphilis may cause congenital abnormalities.
   2. Rubella in early pregnancy may cause: I. Cataract. II. Heart lesions. III. Microcephaly. IV. Deafness[7].

7. Blood incompatibility: Opposite Rh factors in parents may lead to anomalies.

Hereditary Congenital Anomalies:

These are caused by the mutation of the genes. Fused fingers and achondroplasia are examples of dominant mutations. Haemophilia, colour blindnes and albinos are examples of recessive mutations.

Lastly there is the old belief (superstition) that the mother can condition her baby by good habits or spoil her baby by fright, dietary upsets, etc. So far as we can analyse, this seems to be without any foundation, as there are no nervous communication between the mother and the child.

To conclude, we can only say that the congenital anomalies are many, the causative factors we know are few and the probable causative factors which we do not know may be many. Many of the causative factors can combine to cause a single defect. At the same time one causative factor working under different conditions can cause different anomalies. Much research has to be done in this field through experimental embryology.

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CARE AND PRESERVATION OF EYE-SIGHT

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Of the five senses, sight is held to be most precious and the care and preservation of eye-sight is of paramount importance to mankind in all walks of life. Sight makes us acquainted with the world around us; it helps to protect us from external injury, it contributes the largest store of information, it reveals the beauties of colour, shape and form; the treasures of nature; the glories of earth, sky and sea and knowledge through the written word.

Mankind believes implicitely in its eyes, so much so that the phrase "seeing is believing" has become a proverb. The proper use of the eye is, however, an educative process. Objects are perceived by the brain not only as photographed on the retina, but in comparison with one another in relation to colour and form. The infant who tries to grasp the moon with his hand has to learn to appreciate the distance in order to use the eyes properly. Sight is, therefore, a physiological process and not a mechanical one. We learn to see with our brains as well as with our eyes.

The best use is not always made of the eyes. Training does much to help the use of the eyes. The connoisseur sees beauties or imperfections in a work of art which escape the untrained eye. The physician, through his special power of observation and experience, can often diagnose disease merely by looking carefully at the patient. The untrained eye will ignore many objects of interest and importance. So true is the remark "you see, but you do not observe". With all these imperfections, eyes are the most precious gift to mankind. They are a possession to be cherished and trained in order to be of the greatest use to their owner. So every care should be taken to preserve them by proper ocular care and hygiene.

The eyes share in the general nutrition of the body. The different parts of the eye contain and require special chemical substances which are supplied by the diet. Certain vitamins are important for the preservation of good eye sight. Vitamin "A" builds up visual purple in the retina and prevents night-blindness, while lack of "B" vitamins impair the eye sight and may cause blindness. The diet should contain proper amounts of protein, fats, carbohydrates, minerals, adequate amounts of vitamins "A", B_1 and B_2. In a diet deficient in vitamin A, the eye is affected in various ways. The common defect is night blindness, the eyes being unable to adapt themselves to dim light. If the deficiency is prolonged, the conjunctiva shrinks, becomes dry and the condition is termed xerophthalmia. In even more advanced cases, the cornea becomes dry and opaque, ulcers may form on it resulting in permanent scars. The "B" group of vitamins is also of importance. Lack of vitamin "B_1" or Thiamin, causes optic neuritis. Deficiency of vitamin "B_2" or Riboflavin, may give rise to opacities on the cornea. The chief sources of the "B" group of vitamins are whole cereals and pulses, liver and yeast.

Before the birth of the child an expectant mother. if she has any discharge from the genital passages, appropriate treatment is to be given to avoid infection of the baby's eyes at birth. Immediately after the birth, the child's lids should be carefully wiped with damp cotton wool before the eyes are opened. The bath water from the first bath should not be allowed to get into the child's eyes. Any discharge from the child's eye should be immediately treated and any delay might prove dangerous. Care should be taken to avoid the injudicious exposure of the eyes of small infants to direct sun light.

The delicate eyes of growing children should not be subjected to eye-strain. For these reasons it is very important to test the eyes of school children periodically. School age is the period of life when most strain is thrown on the eyes. Realising the particular liability to short sight of children at the school age, teachers, parents and guardians should be on the look out for symptoms of eye strain in children such as head ache, visual fatigue and inability to read letters or distinguish objects at a distance. If the child shows these symptoms, the eyes should be tested and if myopia be be present, spectacles should be prescribed and the child should wear the glasses constantly, because in addition to assisting good definition and relieving eye strain they tend to check the further progress of short sight. Visual defects in school children can be prevented by school work being done under proper conditions of lighting. Good lighting is necessary to prevent eye strain, whether or not it prevents or limits the development of short sight. Bad lighting added to bad eye sight are responsible for much backwardness in school children. Care of the youthful eye is also helped by proper nutrition, by providing well printed books, by holding the book while reading at a proper distance from the eyes and by correcting faulty posture. Desks and seats should be of the proper size for the child. The seats should be of correct height from the floor and be provided with back rest. The desk should be sloped at an angle of about 15° to facilitate writing.

The back edge of the desk should overlap the front edge of the seat. The desk should be adequately illuminated and the light must be evenly distributed. The other point of importance is to avoid glare from the brighter portions. Glare has two bad effects on the eye. It produces a sense of discomfort and also actual disability, for the eye cannot distinguish an object near to a glaring source of light. The best example of these bad effects is the glare of the powerful head lights of a motor car. The school leaving child should receive another eye test to determine if there is any defect of vision which might prevent his further progress in his intended occupation.

In this connection I would like to stress the importance of eugenic prophylaxis in the eradication of high myopia. Prophylaxis is the best form of treatment. Two high myopes should not marry. Instead of astrologers fixing up marriages the family physician has to do the fixing of marriages. By such means a considerable amount of avoidable bad inheritance could be annulled or neutralised. We should only encourage healthy genes to survive and thrive and gradually wipe out defective ones. Such selective breeding will be of great benefit to the individual and the nation. The question is often asked by patients whether

refractive errors could be cured without glasses. There is no basis for the tall claims made by some people in curing these defects without glasses. They of course appeal to the credulous who are, however, soon disillusioned. People should be warned about the danger of placing any faith in them.

The eyes of children require special care during attacks of measles and small pox. All children should be vaccinated and revaccinated against small - pox during epidemics. Smallpox causes ulcers on the cornea of one or both eyes which perforate and lead to loss of the eyes.

Squint appears usually in childhood. The eye that squints always has poor vision. In some cases the squinting eye may be blind. Squint may be brought about by paralysis of a nerve or nerves supplying one or more of the eye muscles or may be due to weakness or over-strain of one or more of these muscles. Squint appears also after infantile disorders such as convulsions, whooping cough, diphtheria, measles, a fright or fall. Squint can be cured and corrected, but due to an old belief that it is a lucky sign especially in a female child, many parents do not like it to be corrected. It is very important to have the treatment as the squinting child on growing suffers from an inferiority complex. Squint can be treated by optical correction, eye exercises and by operation.

During adult life constant care should be taken to protect the eyes in various occupations and employments.

Later in life when the forties and fifties are reached, the onset of presbyopia should not be forgotten. Many go through a period of discomfort before they realise that they require glasses for near work.

The chief affections of the eye in old age are glaucoma and cataract. Care of the eyes in old age is highly important and much can be done to help them to retain or regain their sight. Glaucoma is an affection of the middle and late years. It is a disease that robs the sight without any warning. The sight is lost before one is aware of it. But if detected early and treated, the disease can be checked. Glaucoma is caused by increased pressure inside the eye ball. Normally the eye is soft and tender, but in glaucoma it becomes as hard as a glass marble. This increasing hardness or pressure destroys the optic nerve and sight is lost slowly but surely and the victim is scarcely aware of what is happening.

Cataract, due to opacity of the crystalline lens, causes dimness of vision. As the cataract progresses, the sight becomes worse and at length only a faint perception of light may remain. Removal of cataract by operation is the only cure. With the modern technique, the cataract can be successfully extracted at any stage. Advanced years are no contra-indication and many people over 80 years of age have had their cataracts successfully removed. A general medical examination and an examination of the eye to see that no other affection is present are made before the operation. The delight that the patients express in their ability to see again is a tribute to the success of the operation. It is in this condition quack remedies and operations have written permanent history over the eyes of many victims. The quack performs an unscientific and dangerous operation of "couching"— dislodging the cataract into the eye ball, a dangerous procedure, which although restores some sight

immediately, is always frought with grave consequences eventually leading to blindness.

Eye hazards in industrial workers is of utmost importance. The eyes of the worker bear the brunt of industrial trauma. Eye injuries occur in industry and agriculture and they can be prevented by suitable precautions, as in factories by the use of machine-screens, dust extraction fans and by wearing goggles. Eye injuries are of various degrees of severity. Most of the foreign bodies which enter the eye in industrial processes are hot and thus the injury is not only a wound but also a burn. Small fragments of metal may perforate the cornea and remain in the eye so that there may be very little evidence of the perforation. Hence all apparently slight injuries require careful examination and treatment. All entrants to industry should be examined for visual defects. If the defects are not detected and corrected, they tend to favour industrial accidents. It is also important to detect colour blindness and night blindness in workers. Certain individuals are more subject to accidents than others. Detection of such "accident prone" persons would minimise the risk of industrial accidents and eye injuries. It is desirable to have a periodical examination of eyes of workers. Education of the worker by talks, leaflets and personal advice concerning the risk of eye injuries in this particular industry and precautions necessary to avoid these injuries constitute another important preventive measure. Workers in lead industry sometimes become blind, so also glass-workers exposed to the glare of the furnace, who acquire a special form of cataract. Factories should be properly illuminated as defective illumination is apt to have a serious effect upon vision. Glare and shadows are responsible for eye-strain and consequently diminish the output in factories.

Care should be taken when a foreign body gets into the eye. One is tempted to deal with the foreign body himself or by other unskilled persons with the consequent risk of infection to the eye. If an eye becomes angrily inflamed or if there is any sudden loss of vision, medical aid should be sought at once. Regarding the causes and prevention of blindness, the best way to diminish blindness is to prevent its occurrence. Every conceivable care should be taken to reduce the incidence of eye disease the causes of which are to a large extent preventable and remediable. Innumerable eyes could be saved if only people realise the importance of seeking proper medical aid in the early stage of the disease.

The common causes of blindness are:

1. Ophthalmia neonatorum (sore eyes in newly born babies)
2. Small-pox
3. Venereal disease
4. Trachoma
5. Xerosis and keratomalacia (i.e. eye affections due to vitamin A deficiency)
6. Accidents and injuries
7. Short sight or myopia
8. Squint or cross eye
9. Cataract
10. Glaucoma
11. Quack remedies

Poverty, ignorance, apathy, superstition, malnutrition, absence of facilities of modern medical aid are other contributory causes. Intensive

propaganda among the people in rural areas about the care of the eye would go a long way in eradicating all forms of preventable blindness. Any disease can be stamped out in a comparatively short time if proper approach to solve the problem is made. What is needed is a united effort in the crusade against blindness. Medical science, voluntary effort and administration should all combine to play this vital role. The ever increasing advances in ophthalmic science carry hope and promise of good eyes and eye-sight for every individual. There is no other branch of medical science in which comparatively cheap and speedy treatment yield such quick, satisfactory and lasting results. Vigorous and determined efforts in the right direction are essential to see that none of us shall ever have to go blind in our life time.

STATE PRESIDENT'S LETTER

I am very happy to inform you that at the meeting of the Central Council of the Indian Medical Association held last week at Poona it has been decided that the 39th All India Medical Conference shall be held at MADRAS under the auspices of the Madras State Branch of I. M. A. Our State is, thus, having the opportunity of playing the host to the parent body of our association after a very long interval. The conference proper and the meetings of the central working committee and the central council preceding and succeeding the conference, will all come off during the last week of December 1963; and we shall be having the pleasure of the company of veteran members of the I. M. A. and represntatives of all branches of the I. M. A. throughout our country on the occasion. We shall be having an exhibition of medical and pharmaceutical appliances and products; and a good scientific fare during the scientific sessions. An attractive souvenir will also be brought out to commemorate the occasion. The work connected with all these can successfully be completed only with the generous, active and kind co-operation of all the members of I. M. A. in our state and office-bearers of all the local branches in our state in particular. As the president of this State branch of I. M. A. and as the person responsible for organising this great conference I hereby appeal to you and through you to all the members of I. M. A. in your jurisdiction to kindly extend your full co-operation, help and advice to me, and do your valuable bit of service to make this conference a grand success. It shall be our aim to keep up - if not excel - the traditions established by the other branches of I. M. A. who had, so far, conducted the conference; and this will be possible only with your valuable co-operation.

C. NATHAMUNI NAIDU,
President.

PRINCIPLES IN MANAGEMENT OF INJURIES OF HAND *

N. S. K. SWAMY, M. B. B. S., M. S., Erode.

INTRODUCTION :

Art is a conception of the mind, dexterously executed by the limbs and voice. The role of the hands in the creation of art is unique. Music, dance, architecture, painting, science and philosophy, etc., are all created by the superior mind and skilful hands. Life is not worth living but for these wonderful things in this world.

Man became superior to the other species by his mind and hands, which created art, science and philosophy. The role played by the hands is immeasurable and unique in all these things. Thus the importance of human hand. Hence surgery of the hand should receive special attention, especially in these days of modern civilization with rapid industrialisation where human hands are exposed to injury very frequently.

ANATOMICAL FEATURES :

The human hand is an instrument of perfection. The anatomical features are so perfect that no mechanical device is capable of substituting for the hands.

The nails, provided they are kept clean and trim, will be a very useful instrument in picking small objects from a smooth surface. The nail bed has a rich capillary net work which could be used to assess the terminal circulation.

The skin is applied to the fingers so perfectly that flexion and extension does not produce discomfort. It is like perfect gloves which has not been made so far. Multiple transverse ridges in the skin ensure a good fit in all positions. The dorsal skin is thin and freely mobile. The palmar skin is thick, tough, inelastic and fixed in places. Creases and folds allow mobility in the joints. The subcutaneous fat is compressed by fibrous stoma. The flexor area is designed to withstand great pressure but yet very sensitive.

The palmar fascia protects the deep structures and prevents hyperextension of the fingers.

The flexor tendons, short flexor muscles which lie between them and the neuro-vascular bundles lie deeper to the palmar fascia. Bow stringing of the tendons are prevented by annular ligaments attached to the phalanges. Blood supply of the tendon is not affected in any injury. The middle metacarpal is comparitively rigid, whereas the other metacarpals are mobile. The resting hand forms a long arch from wrist to finger tips and a short arch across the heads of the metacarpals, both concave towards the volar surface. All fingers on flexion point towards the base of the thenar eminence. This is the position in which the fingers should be immobilised. The thumb is immobilised in the opposed

* Lecture delivered at a meeting of the Erode Medical Association held on 24—11—1962.

position. Thus the position of function is the tumbler holding position.

Injuries :

These will be classified in the following ways :—

1. Traumatic, thermal or chemical
2. Superficial or deep
3. Open injuries (with dissolution of continuity in the skin surface), closed injuries (without dissolution of continuity of the skin surface).
4. Injuries associated with fracture and injuries without fracture.
5. Combinations of any of the above.

In burns, the main problem is contractures due to scars. Initial treatment is the same as for burns elsewhere.

Principles of Treatment of the Injured Hand :

The aims of treatment should be to obtain first intention healing wherever possible, to restore the normal functioning of the hand ; to prevent infection thus minimising restriction of movements by scars and stiffness of joints.

Bleeding is controlled by compression and clean firm bandage as first aid is done. Elevation of the limb or, if necessary, a temporary splint is given.

The definitive treatment in the hospital is first directed towards combating shock and treatment of general diseases, if any. All severe injuries of hand are better treated as inpatient.

Diagnosis :

By history :— The exact mode of injury, cut or crush is recorded.

By clinical examination :— Local examination will reveal the extent of damage. General examination for associated injuries and general diseases.

X-rays : To locate foreign bodies, fractures and dislocations. By a thorough examination, the entire picture is recorded. Damage to all the structures including the neuro-vascular bundle is assessed and recorded. It is important to record the occupation which is important for the rehabilitation of the patient.

Anaesthesia :— Local or brachial block is quite suitable.

Anatomical Exposure :— The enlargement of the wound is done, whenever necessary, along the creases avoiding crossing the joints. In the fingers the incisions are made over the sides and not over the palmar or dorsal aspects.

Control of Haemorrhage :— Tourniquet is better avoided, because tissues with meagre blood supply may die and it may be difficult to distinguish dead from living tissues while excising the wound. Pneumatic tourniquet is universally recommended, if it is absolutely necessary to use a tourniquet. Small bleeding points are controlled by pressure and large bleeding vessels are ligated. The minimum amount of foreign material is buried the better. Rarely ligation of the artery is required to control the haemorrhage. If it is necessary, ligation of the brachical artery only will control the bleeding and not the radial and ulnar arteries.

Wound Excision :

The excision of a wound done carefully and properly will fulfil major requirements for a good healing and restoration of function.

The following principles are followed:

1. Removal of dead tissue.
2. Removal of foreign bodies.
3. Secure a free bleeding edge from the wound after excision.
4. Excision is done layer by layer.
5. After haemostasis the wound can be sutured without a drainage if excision is satisfactory.

Tendons are not repaired primarily except when all facilities are available and the wound is clean. It is necessary to remember the anatomical points regarding the tendons of the palm. Palmaris longus is subcutaneous. Next is the tendon of the sublimis. Deeper to it is the tendon of the profundus.

No two tendons are sutured at the same level for fear of cross adhesion thereby impairing the functional efficiency of the hand. The tendon suture should not be opposite a skin suture for the same reason. If both the flexor tendons are injured, the sublimis is removed and the profundus is repaired. For bridging any gap the sublimis tendon or palmaris longus could be utilised. The tendon sheaths are repaired carefully for smooth gliding of the tendons in action.

Nerve: Digital nerves do not regenerate unless repaired properly. Only the perineurium is sutured.

Other general principles: Whenever there is skin loss, skin cover is provided by skin grafting, whole thickness or pedicle graft. The skin of the thenar eminence is well suited for the replacement of the pulp of the fingers. In a few cases I have done, the results are gratifying. Extensive loss of skin is made good by pedicle grafts from the abdomen or the thigh. The hand could be buried in the abdominal wall or the thigh in certain circumstances and flaps are fashioned later as needed.

Amputation of the digits are avoided as far as possible. The thumb is seldom amputated, for even a rigid thumb is useful. If the thumb is lost inevitably, ingenious plastic surgery techniques are needed to create a thumb. Example: Rotation of the first metacarpel to the position of the thumb.

Management of Late Cases:

If more than six hours have elapsed, excise the wound, but no suturing is done. If no sepsis occurs, secondary suture is done.

Management of Early Cases:

If the patient is brought within six hours after injury, the following procedures are adopted:

(1) Incised wounds: Excision and suture.

(2) Contused wounds: Excision as suited for individual cases. If there is a gap, avoid tension in suturing. Skin grafting is done if necessary.

(3) Penetrating wounds: Entry wound may be small. Always examination is made for tendon or nerve injuries, wound excision is done, enlarging the wound and exploring the depths and then the wound is sutured.

(4) If there is a haematoma without skin loss, it is let out, thus avoiding a nidus for infection.

(5) Loss of skin: The best dressing for the wound is skin. Partial thickness skin graft is good. The graft should not be placed on the tendons, bone or capsule of a joint. Vaseline

gauze or tulle grass dressing and pressure bandage is necessary after grafting. A suitable splint in position of function is given. Successful skin grafting quickens the healing, facilitates further surgical procedures as tendon or nerve repair.

(6) Replacement of completely severed portions of digits :– If the patient is brought within half an hour, the severed portion is sutured in position after washing it in saline. After 48 hours, if it sloughs, it is removed. In one case I have treated, where a suturing was done by a private practitioner a few months ago, there was an encircling scar of the thumb except a $\frac{1}{2}$ cm. area over the medial aspect. There was a complete fracture of the proximal phalanx with infection and a small raw area over the lateral aspects, the bone forming the base of the wound. After four months of persistant treatment of infection and adopting minor surgical procedures like scrapping, etc., a complete healing and restoration of the thumb with some restriction of movements was obtained.

(7) Closed injures : The hand is kept in the position of function and dressed with plenty of cotton wool. The limb is kept elevated. Cortisone treatment is advised in such cases. Prednisolone is given 40 mgs. for the first 5 days, 30 mgs. for the next five days and 20 mgs. for the last five days (Hamilton Bailey).

General Principles in Treatment of Fractures and Dislocations :

(1) Accurate reduction and immobilisation.

(2) Immobilisation is done in a semiflexed position except rarely, the tip of the finger pointing towards the base of the thenar eminence. A stiff finger in this position is useful, whereas a stiff straight finger is a hindrance to the normal functioning of the hand.

(3) Immobilisation of the fingers are done for not more than three weeks after which active movements should be started.

(4) No massages or forced movements are done at any stage. Movements are regained only by persistant active movements.

(5) Fingers other than the immobilised one are exercised from the very first day.

(6) Compound fractures are treated by wound excision and suture. The fracture is reduced and immobilised as for a closed injury.

CONCLUSION :

It is worth while to remember that surgery of the hand injuries are more important, much simpler, less expensive than the more dramatic intracranial surgery, etc., and yet leaves a fitter individual. But for proper treatment of the hand injuries, the individual may be greately handicapped.

Economically prevention and treatment of hand injuries are far more important. Preventive measures in industries with the close co-operation of the industrial heads will benefit the patients much. With the creation of more and more machinery we expose our hands frequently to accidental injuries. Well organized 'Hand Surgery Clinics' at least in the major hospitals are essential. "Save the Hands" will be an appropriate slogan at this juncture.

ABSTRACTS AND EXCERPTS

"FOLIDOL" (Parathion) POISONING:

In the Government General Hospital at Jaffna (Ceylon), there were 62 cases of folidol poisoning seen over a six-month period from January to June, 1960: 51 accidental and 11 suicides; 9 of the 62 proved fatal. It is rather alarming that during this period of study folidol accounted for 72% of all cases admitted to this hospital for poisoning and for 93% of all deaths from poisoning over this period.

Mild cases of poisoning complained only of headache and vertigo. Sometimes they also complained of nausea, vomiting, abdominal pain, paraesthesia, and muscle cramps. Three cases in this series had pain in the cervical region travelling down the spine. In severe cases of poisoning the patient was comatose, with pinpoint contraction of the pupils, which failed to react to light. Convulsions also occured in three cases in this series. When death occured it was due to pulmonary oedema which developed and progressed with lightning speed.

Until medical assistance becomes available, patients with folidol poisoning must be kept at rest and in the fresh air. The antidote for folidol poisoning is atropine. It is administered in a dose of 1 mg. intramuscularly, and may be repeated several times, depending on the condition of the patient every 5 to 15 minutes. In severe cases with miosis and pulmonary oedema 1 mg. atropine may be given intravenously every five minutes, and, disregarding the usual pharmacological maximum dose of atropine, until the miosis just begins to disappear. In this series many required 15 to 17 such doses before the miosis began to disappear, at which stage also the pulmonary oedema was also nearly always brought under control.

Accessory methods of treating pulmonary oedema such as propping up the patient on a bed rest, oxygen inhalations, and mersalyl 2 ml. intramuscularly are equally important. Where the patient is gravely ill, the mersalyl should be given intravenously — two 1 ml. injections at an interval of 15 minutes.

One case in this series developed signs of atropine poisoning, consisting of dry mouth, intense thirst with difficulty in talking and swallowing, dry flushed skin, blurred vision with dilated fixed pupils, and rise of temperature; also a state of excitement with disorientation, hallucinations, and delirium. These symptoms disappeared within six hours of the discontinuance of atropine. Atropine poisoning can be prevented by stopping the drugs immediately the miosis begins to disappear.

Appended below is a list of the minimum precautions required when handling folidol:

1. No one with cuts, abrasions, or ulceration of the skin should handle folidol either for mixing or spraying.

2. An individual should not take part in spraying the insecticide for more than four hours in any one day.
3. Spraying should not be done against the direction of the wind.
4. Clogged nozzles should not be blown out with the mouth.
5. There should be no eating, chewing, or smoking while spraying and until after the clothing worn during spraying has been changed and hands washed with soap and water.
6. Immediately after spraying clothing worn as well as all exposed parts of the body should be washed in soap and water.
7. Spoons and containers used for measuring the liquid should be washed in hot soda solution.
8. Vegetables and fodder can only be considered fit for consumption a week, and fruits two to three weeks, after the last spray. Animals should be kept away from sprayed pastures for at least five days.

— *A. V. A. Vethanayagam, "British Medical Journal; October 13, 1962: Pages 986 & 987."*

* * * *

THE LONG TERM PROGNOSIS OF TUBERCULOUS MENINGITIS IN CHILDREN:

From 1947 up to the end of 1955, 170 children were treated for tuberculous meningitis by the Department of Child Health of the University of Sheffield. One hundred of these survived for 5 years or more. After their discharge from hospital they were all followed at regular intervals at a special follow-up clinic. In the follow-up period much stress was laid on the assessment of their personality, their achievement, and their adjustment to life. Routine independent intelligence tests were performed at approximately annual intervals until a more or less stable result was obtained. Major changes in the test results were only obtained in children who were under 3 years of age on admission. The children underwent routine developmental and neurological examinations which included a study of the fundi, electroencephalography, electric audiometry and repeated x-rays of the chest and the skull. It is very fortunate that these tests could be performed on almost all children.

Results:

Seventy-seven survivors are subjectively normal and 23 have various physical or mental sequelae.

TABLE

The physical condition of 100 children at the last assessment.

Subjectively normal	77	Fits	5
Deaf	4	Facial weakness	1
Blind	1	Gross or combined defects	12

Intelligence:

Six children became profoundly retarded by their illness. All those were under two years on admission. The range of the I. Q. in the remainder is about the same as that of the general population, although the average I. Q. of 94 was below that of the general population. As the composition of the socio-economic status of their families show a heavy preponderance of semiskilled or unskilled workers, this was to be expected. Seven children have an I. Q. of 120 or more. All these were fully conscious on admission and throughout their treatment. In the large majority the meningitis did not cause a detectable loss of intelligence. There is some evidence, however, that six children, whose I. Q. is within the normal range, did suffer some intellectual deterioration as a result of their illness. Among the 12 children who survived with major neurological or endocrine sequelae, there are 6 whose I. Q. is within normal range.

Detailed information is available about the scholastic progress of all the children who attended schools after their meningitis. Broadly speaking and within their appropriate environment about one-third were making excellent progress, and a third were of average ability; another third were dull and making poor progress.

A correlation between I. Q. and scholastic achievement was remarkably consistent. Only 5 were not doing as well and 2 were doing better than one might have expected from their I. Q.

Neurological lesions and other physical defects:

All the children who showed definite intellectual deterioration also have neurological sequelae. There are altogether 23 children who had neurological sequelae, including isolated deafness, and who had subjective disability as a result of these sequelae. There were, however, several other children who did have gross neurological lesions during the active phase of their disease but who made a complete recovery.

Hearing:

Eighty-four children have normal hearing, including 3 children who had been deaf for a long period after their recovery. Two of them were of school age and had actually attended a deaf school for a period. Their hearing gradually returned and all 3 now attend ordinary schools without difficulty. Four children are completely deaf.

Vision and ophthalmological aspects:

There were 3 blind children and all 3 had complete optic atrophy. Two of these are aments with multiple neurological defects and their blindness is of no clinical importance. One child, however, was perfectly normal in every other way and was a good scholar in a blind school. He had extensive intracranial calcifications at the site of the chiasma. There were 4 other patients whose optic atrophy was and remained complete as

judged by ophthalmoscopic examination, yet their vision is normal both subjectively and on expert testing of visual acuity. There are 3 children who recovered completely from total blindness associated with papilloedema and 2 who recovered from partial blindness.

Choroidal tubercles were still present at the last examination in 31 children in whom they were found at the beginning of their illness. In the latest and presumably permanent stages they appear as dead white scars with either a moderate amount of black pigment scattered largely around the periphery, or with masses of black pigment almost completely covering it.

Convulsions:

Convulsions persisted after recovery in 8 children. Among these are 5 whose fits are their only sequelae and which are well controlled by drug therapy.

Paralytic lesion and endocrine sequelae:

There are 12 survivors with paralytic manifestations of various types, often associated with other features which have been described already. Seven suffer from spastic hemiplegia of varying severity, and 1 of these has residual paraplegia as well. There are 2 children with residual paraplegia of moderate degree and one child has spastic quadriplegia. There remain 2 children whose motor defect is of lower motor neurone type. It is very likely that one of these had an associated poliomyelitis.

It is noteworthy that the I. Q. of 6 of the severely paralysed children is below 59, 4 others have an I. Q. between 75 and 90, and only 1 has an I. Q. as high as 100.

An unusual source of disability was the occurrence of bilateral ectopic ossifications of the iliopsoas tendons and in the adductors of the thighs. This occurred in 3 survivors. All were in the advanced stage on admission and all had at one time paraplegia in flexion.

The only obvious and gross endocrine disorder which was encountered in this series was sexual precocity. All 3 are girls, all 3 were in the advanced stage on admission, all developed gross hydrocephalus, and all had or still have gross neurological lesions.

The other very rare endocrine disturbance which has been described is diabetes insipidus. There have been no such case in this series, but I investigated 5 boys because of gross polyuria and polydipsia following tuberculous meningitis. Pitressin-sensitive diabetes insipidus was confirmed and they are doing well on treatment.

Intracranial calcifications:

An interesting feature of children who recovered from tuberculous meningitis is the high incidence of intracranial calcifications which are found in 45 children. These calcifications most commonly became demonstrable

between 2 and 3 years after the onset of the disease, but occasionally they were not seen for up to 5 years. These calcifications are commonest in the basal meninges and much less common in the substance of the brain itself. Very gross calcified masses may be seen on radiographs of the skull of perfectly normal children. Often there is no calcification at all in children with the most extensive neurological damage.

It is concluded that with earlier diagnosis and with modern treatment the incidence of serious sequelae from tuberculous meningitis could well be reduced to negligible levels.

—'*Dr. J. Lorber, University of Sheffield.*'
(*"Pediatrics", 1961,* 28 : *778.*)

EXTENT OF SPURIOUS DRUGS IN THE COUNTRY

The Government of India, Ministry of Health have appointed the Drugs and Equipment Standards Committee under the chairmanship of Dr. D. S. Raju, Deputy Health Minister, charged with the duty of, among other things, assessment of the spurious as well as substandard drugs in the market. In the interests of the indigenous drugs industry and in order to safeguard the health of the people it is desirable to have a fairly accurate assessment of the extent of spurious drugs in market. The committee, therefore, requests the full co-operation of the medical profession in making available to it the following information :— (1) The types of spurious drugs that the members of our association / profession have come across in the course of professional work or otherwise ; and (2) particulars of such drugs including the name of the drugs, the name of the manufacturer as shown on the label.

In replying to these queries it will be appreciated if the distinction between spurious and sub-standard drugs is borne in mind. Spurious drugs will fall under the following categories :

(a) A drug whose label shows it to be manufactured by a firm which is non-existent.

(b) A drug which is found to be different from what is claimed on the label.

(c) A drug which is manufactured by a party other than manufacturer shown on the label.

(d) A drug which is a close colourable imitation of a well established drug or brand of drugs and which is likely to deceive the consumer into the belief that he is buying the established drug or brand of the drug.

(e) Defective drugs which are treated in such a manner as to conceal the damage or defect of drugs which are made to appear of better or greater therapeutic value than they really are. (Penicillin adulterated with other material and labelled as pure penicillin of certain potency is a case in point).

The information on this subject that may already be in possession of members or such information as the members may be able to gather now, may kindly be transmitted directly to "Dr. S. K. Borkar, Member-Secretary, Drugs and Equipment Standards Committee, Directorate-General of Health Services, NEW DELHI", without delay.

NATIONAL DEFENCE FUND — DONATIONS THROUGH THE MADRAS STATE BRANCH, I. M. A.

The following donations to the National Defence Fund received through the honorary secretary of the Salem branch of the I. M. A. are gratefully acknowledged:

			Rs.	nP.
1.	Dr.	K. Jayaramachandran, Namakkal	50	00
2.	„	Kumari B. Mukambu, Namakkal	13	00
3.	„	Shiva Shanker Bolar, Salem	10	00
4.	„	A. S. Krishnn Iyer, Salem	10	00
5.	„	M. V. Sethumadhavan, Salem	10	00
6.	„	C. Nachiappan, Salem	10	00
7.	„	J. Sugavanam, Salem	10	00
8.	„	B. L. J. Mathuram, Salem	10	00
9.	„	M. Devanesan, Salem	10	00
10.	„	K. N. Padmavathy, Salem	10	00
11.	„	Mrs. M. Panchanadam, Namakkal	10	00
12.	„	Maragadam Mariappan, Salem	10	00
13.	„	K. Subbulakshmi, Salem	10	00
14.	„	U. B. Lakshmi, Salem	10	00
15.	„	A. J. Arunagiri, Salem	10	00
16.	„	K. Ramanathan, Salem	10	00
17.	„	K. Govindarajulu, Salem	10	00
18.	„	P. D. Bhushanam, Namakkal	10	00
19.	„	Ramachandranaick Sujir, Salem	10	00
20.	„	K. C. Roy, Salem	10	00
21.	„	B. Mari Chetty, Salem	10	00
22.	„	C. Chinnakrishnan, (Sympathiser)	10	00
23.	„	R. Venkatappa, Salem	10	00
24.	„	M. R. Srinivasan, Salem	10	00
25.	„	K. N. Rao, Salem	10	00
26.	„	L. S. Natarajan, Dharmapuri	10	00
27.	„	A. P. K. Nair, Salem	10	00
28.	„	S. Muthuswami, Salem	10	00
29.	„	Capt. S. N. Venkataraman, Salem	10	00
30.	„	G. R. Rajaram, Salem	10	00
31.	„	Capt. V. Raghavendra Rao, Salem	5	00
32.	„	T. S. Kalkura, Hosur	2	00
		Total ...	350	00

ASSOCIATION NOTES

BRANCH NOTES

Chettinad Branch:

The annual meeting of the Chettinad branch, Indian Medical Association was held on Saturday, the 10th November 1962 in the N. S. M. V. P. High School, Devakottai, Ramnad District.

At a business meeting the following office-bearers for the year 1962—1963 were duly elected.

President	:	Dr. A. Vaidyanathan
Vice-President	:	Dr. Mrs. L. Sukumar
Hony. Secretary and Treasurer	:	Dr. (Capt.) G. D. Ebenezer
Hony. Joint Secretary	:	Dr. P. K. Narayanan
Committee Members	:	Dr. (Major) T. D. Gowrisanker
		Dr. T. V. Ramanan
Representative to State Council	:	Dr. S. Subramaniam
Representative to Central Council	:	Dr. A. Rahman
Auditor	:	Dr. K. Raghavan

There were two lectures by the following doctors:

1. Dr. P. K. Kalyanaraman, M. B., B. S., Physician, Coimbatore on 'Refractory Heart Failure'.

2. Dr. O. Francis, M. B., B. S., D. G. O, M. D., Professor of Obstetrics and Gynaecology, Madurai Medical College, Obstetrician and Gynaecologist, Government Erskine Hospital, Madurai on 'Intranatal Care'.

The following films were shown by the courtesy of M/s. Sandoz Ltd.:

(i) Inter-Atrial Septal Defect and its Repair

(ii) Cine-Endoscopy

(iii) X-ray Cinematography

(iv) Swiss Dhanalagiri — Himalaya Expedition.

Chingleput Branch:

At the general body meeting held on Saturday, the 10th November 1962 at 2-30 p. m. at the Government Head Quarters Hospital, Chingleput, the following office bearers were elected unanimously for the year 1962–'63.

President	:	Dr. M. Narayanan
Vice-President	:	Dr. P. K. Varghese, Tambaram.
Secretary and Treasurer	:	Dr. A. Sundararaja Rao, Chingleput.

Members of the Executive Committee:

1. Dr. K. V. Rajagopalan, Kancheepuram.
2. Dr. A. N. Jagadesan, Tambaram.
3. Dr. Vittal Doss Pai, Pallavaram.
4. Dr. T. Thayumanava Mudaliar, Kancheepuram.

Representative to the Central Council:

Dr. P. K. Varghese, Tambaram.

Representatives to the Provincial Council.

1. Secretary
2. Dr. K. V. Rajagopalan, Kancheepuram.
3. Dr. A. Rajagopalan, Kancheepuram.
4. Dr. P. S. Ananta Narayanan, Kancheepuram.
5. Dr. C. S. V. Rajappa, Kancheepuram.

After tea, Dr. Ratnavelu Subramaniam, M. R. C. P. (Lond.) dealt exhaustively on 'Congenital Heart Diseases and the Importance of Surgery'. There was a demonstration of films on 'Indications for Siquil' and 'Cancer and its Diagnosis', by the kind courtesy of M/s. Squibbs.

Coimbatore Branch:

The monthly meeting of the association was held at the association premises at 6 P.M. on 8th October 1962. Dr. Mrs. Anna Vareed, the president took the chair and commenced the proceedings. The president congratulated Dr. G. T. Gopalakrishna Naidu on his election as the senior Vice-President of the Madras State Branch of the I. M. A. for the year 1962—1963.

Welcoming the three speakers of the day, the president complimented, them for their young age and talents and requested Dr. Mrs. Lalitha Kameswaran, M. B., B. S., Ph. D. (Lond), Professor of Pharmacology, Madurai Medical College, Madurai, to address the meeting first. The speaker gave an account of the pharmacology and the therapeutic usefulness of some of the drugs sold in the market for the treatment of cough. She dealt at length on the pharmaco-physiological mechanism of cough and their relief for suppression. She explained the use of the drugs with the aid of slide charts.

Dr. Durairaj, M. S., F. R. A. C. S., Asst. Professor of Pediatric Surgery, Madurai Medical College, Madurai next spoke on 'Respiratory Obstruction in the New-born and Infancy' with the aid of slides, the speaker cited the various cases of respiratory obstruction in children and their treatment.

Dr. S. Kameswaran, M. S., F. R. C. S., F. R. F. P. S., D. L. O., Assistant Professor of Otorhinoloryngology, Madurai Medical College, Madurai, spoke on 'Common Respiratory Emergencies in Childhood', made some observations on the merits of the practice as he saw in U. K. and in our country, substantiating his talk with statistics.

The speeches were very informative and interesting and the members evinced a keen interest by putting questions at the end of every talk.

Erode Branch:

Dr. M. N. Shenoy presiding, a meeting of the Erode Medical Association was held on 24th of November, '62.

Dr. N. S. K. Swamy, M. B., B. S., M. S. Orthopaedic Specialist, Erode, spoke on "Injuries of Hand." 20 doctors attended. An on-the-spot collection for Defence Fund was made. An amount of Rs. 757/- was realised and was sent to the Honorary State Secretary, I. M. A., Madras State Branch pursuant to the instructions received from the President of the State Branch.

Madurai Branch:

A monthly meeting of the Madura Medical Association was held on Saturday, the 10th November 1962 under the presidentship of Dr. K. Ramachandran, M. S., Madurai. Dr. T. Dorairajan, M. S., F. R. A. C. S., Assistant Paediatric Surgeon, Erskine Hospital and Assistant Professor of Surgery, Madurai Medical College, Madurai gave an interesting lecture on 'Urological Problems in Childhood.'

Pudukottai Branch:

The annual meeting of the association was held on 11—11—1962 at 4 P. M. with Dr. C. R. Thiruvengadam in the chair. Fifteen members and guests were present. The following were elected as office-bearers for the ensuing year:

President	:	Dr. C. R. Thiruvengadam
Vice-President	:	Dr. R. Raghunathan
Secretary and Treasurer	:	Dr. N. Thiagarajan
Representative to the State Council	:	Dr. Nachiappan

Dr. Jayakumar, M. D., Prof. of Medicine, Tanjore Medical College, Tanjore gave a very forthright and candid lecture 'Essentials of Medicine'.

Dr. C. N. Santhanam, M. S., Coimbatore gave an interesting lecture on 'Surgical Emergencies in the Neo-Natal Period' with illustrations.

Ramnad Branch:

A meeting of the Ramanathapuram District Branch of I. M. A. was held on Sunday, the 11th November 1962 at 5 P. M. in the 'Kamak Hall' of S. H. N. V. High School, Sivakasi. Dr. S. Raju Ayyar, L. M. P., presided over the function. Dr. C. Jeyaraj Aiya Nadar, M. B., B. S., D. C. H., A. B., Paediatrics (U. S. A.), Pediatric Physican, Erskine Hospital, Madurai gave a very interesting lecture on 'Some Common Ailments in Children'. He covered most of the subject by showing charts and pictures and clinical photographs.

2. A meeting of the Ramnad District Branch of I. M. A. was held on Sunday, the 16th December 1962 at 5 P. M. in the Hindu Nadars' Higher Elementary School, Aruppukottai. Dr. S. Raju Ayyar, L. M. P., President presided over the function. Dr. K. A. Kalyanam, F. R. C. S. (E), D. M. R., Madurai Medical College, Madurai gave a very interesting and useful lecture on 'Some Common Surgical Problems'. He also showed a few slides.

Salem Branch:

A meeting of the Indian Medical Association, Salem Branch was held at 6 P. M. on Sunday, the 9th December 1962 at 'Hotel Dwaraka' Salem. Dr. K. Jayaramachandran presided.

Dr. R. Sarat Chandra, M. S., F. R. C. S., addressed the members on 'Non-acute Surgical Abdominal Conditions'.

A sum of Rs. 350/- was collected from the members present for the National Defence Fund to be sent through the Madras State Branch of I. M. A. in response to the appeal issued by the president.

Tiruchy Branch:

The 33rd annual meeting of the association was held on Sunday, the 11th November 1962 at the medical association premises, Tiruchy. Dr. T. V. Ranganathan, the president presided. After breakfast, the morning session began with a prayer.

The following office-bearers were elected unanimously for the year 1962—1963.

President	:	Dr. C. Ramanujachary
Vice-President	:	Dr. T. V. Sundaram
Honorary Secretary	:	Dr. N. V. Muthukrishnan
Joint Secretary and Treasurer	:	Dr. M. V. A. Iswaran
Executive Committee	:	Dr. A. Dharmarajan
		,, V. Enok
		,, M. A. R. Shakir
		,, P. Palaniyandi Pillai
		,, S. Venkatesan
		,, Mrs. Visalakshi Krishnamurthy
		,, T. Venkataraman
State Council Representatives	:	Dr. T. V. Srinivasan
		,, V. K. Ranganathan
		,, S. Venkatesan
		,, R. Narasimhan
		,, V. N. Lakshminarayanan
		,, V. Enok
		,, N. V. Muthukrishnan, Ex-officio

Central Council Representatives: Dr. T. V. Srinivasan
„ N. C. Subramanyam

Then the new president, Dr. C. Ramanujachary took the chair. After expressing his thanks for electing him as the president for the year, he introduced Dr. C. R. R. Pillay, M. D., M. R. C. P., Honorary Professor of Therapeutics, Stanley Medical College, Consulting and Honorary Physician, Stanley Hospital, Madras to the members and requested him to give his talk on 'Cardiac Neurosis'. After lunch kindly given by M/s. International Chemical & Biological Institute Private Ltd., Bangalore, the afternoon session commenced at 2-30 P. M. Dr. R. Subramanyam, B. Sc., M. D., M. R. C. P., Professor of Medicine, Madras Medical College and Physician, Government General Hospital, Madras addressed the members on 'Congenital Heart Diseases in General Practice'.

Then Dr. A. A. Asirvatham, B. A., M. S., F. R. C. S., Professor of Surgery, Madurai Medical College, Surgeon, Government Erskine Hospital, Madurai addressed the members on "Early Diagnosis in Surgery".

All the lectures were very much appreciated by the members.

After tea by M/s. Professor Gajjar's Standard Chemical Works Ltd., Bombay, a number of medical films were shown to the members by M/s. Sandoz India Ltd., and they were very much appreciated. After a sumptuous dinner by M/s. Sandoz India Ltd., the grand function came to a close at 9-00 P. M.
